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Health and Environmental Effects Profile for Ethyl Methacrylate

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16. ABSTRACT

The Health and Environmental Effects Profile for Ethyl Methacrylate was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA) and to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Ethyl methacrylate has been determined to be a systemic toxicant. An Acceptable Daily Intake (ADI), defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect, for ethyl methacrylate is 0.086 mg/kg/day for oral exposure.

The Reportable Quantity (RQ) value of 1, 10, 100, 1000 or 5000 pounds is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified by CERCLA based on chronic toxicity. Existing data are insufficient to determine an RQ value.

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR ETHYL METHACRYLATE

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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched."

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3112. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

ACKNOWLEDGEMENTS

The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3228 for U.S. EPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa was the Technical Project Monitor and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Solid Waste, Washington, DC.

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EXECUTIVE SUMMARY

Ethyl methacrylate is a colorless liquid with an acrid acrylate odor (U.S. EPA, 1985a). It is chemically reactive and can be readily polymerized (Nemec and Kirch, 1981; Hawley, 1981). The primary use of ethyl methacrylate is for the production of acrylic polymers used in building automotive, aerospace and furniture components and in the manufacture of paint and coatings (U.S. EPA, 1985a). Ethyl methacrylate polymers are also used for making dentures (Freeman, 1965), contact lenses (Refojo, 1979) and artificial fingernails (Lee and Orlowski, 1976). Domestic production figures are not available for ethyl methacrylate; however, its uses suggest that multimillion pound quantities are produced annually. DuPont (Belle, WV) is the sole U.S. manufacturer (SRI, 1985). In 1977, Rohm and Haas Co. imported between 1 and 10 million pounds of ethyl methacrylate (U.S. EPA, 1977).

Experimental data pertaining to the environmental fate processes for ethyl methacrylate were not found; therefore, its environmental fate has been estimated from its physical properties, chemical structure and by analogy to the fate of similarly-structured compounds. In water, hydrolysis, direct photolysis, adsorption and bioaccumulation are not expected to be important. Volatilization from water is expected to be significant, based on the calculated Henry's Law Constant. Ethyl, methyl and butyl acrylate, as well as methyl methacrylate, have been shown to undergo significant biodegradation (Sasaki, 1978; Price et al., 1974; Ekhina and Ampleeva, 1977). By analogy, ethyl methacrylate is expected to be biodegradable. Once released to the atmosphere, ethyl methacrylate is expected to react rapidly with hydroxyl radicals and ozone, with a calculated half-life of ~4 hours (U.S. EPA, 1985b). Ethyl methacrylate is estimated to be

moderately to highly mobile in soil, and therefore susceptible to significant leaching. It may also volatilize significantly from soil surfaces.

Monitoring data regarding exposure to ethyl methacrylate through food, dermal exposure, ambient water or ambient air were not located in the available literature. Ethyl methacrylate may be emitted to the atmosphere or to water from discharges generated during its industrial manufacture or use. Leaching of the unreacted monomer from polymers made from ethyl methacrylate may be a potential source of exposure to either groundwaters beneath landfills that contain the polymers, or to foods packaged in the polymer products. Incineration of polydimethacrylates may also release ethyl methacrylate into the atmosphere.

Pertinent data regarding the carcinogenicity, reproductive effects, subchronic or chronic effects of ethyl methacrylate could not be located in the available literature as cited in the Appendix. Ethyl methacrylate was not mutagenic in Salmonella typhimurium with or without metabolic activation (Waegemaekers and Bensink, 1984). Ethyl methacrylate caused a significantly increased incidence in gross abnormalities after intraperitoneal administration in rats at all dose levels tested (Singh et al., 1972). Acute oral exposure to ethyl methacrylate resulted in an increased and then decreased breathing rate, dyspnea, coma and death from respiratory failure (Deichman, 1941).

The only information about aquatic toxicity of ethyl methacrylate was provided by Bringmann et al. (1980), who reported a toxicity threshold of 178 mg/l for Chilomonas paramecium.

Since there are insufficient data on ethyl methacrylate with which to derive an ADI, an ADI for ethyl methacrylate can be derived based on analogy to the chronic toxicity of methyl methacrylate, a compound similar in structure and toxicity. Because of the consistent teratogenic effects observed after intraperitoneal treatment with ethyl methacrylate and methyl methacrylate, and because teratogenicity is a sensitive endpoint of methyl methacrylate toxicity, the critical effect in determining safe exposure levels to ethyl methacrylate may also be the teratogenic effect. Based on the ADI of 0.075 mg/kg/day for methyl methacrylate, which is protective against the teratogenic effects of methyl methacrylate observed in rats after inhalation exposure, an ADI of 0.086 mg/kg/day for ethyl methacrylate was derived by correcting for molecular weight differences which may protect against the teratogenic effects of ethyl methacrylate as well. This ADI should be considered provisional until subchronic or chronic oral toxicity and oral teratogenicity studies are performed.

The lack of oral or inhalation studies regarding the teratogenicity and reproductive or systemic effects of ethyl methacrylate after either subchronic or chronic exposure precludes derivation of an RQ. The lack of carcinogenicity data precludes the derivation of a potency factor and classifies ethyl methacrylate as a CAG Group D compound (U.S. EPA, 1984).

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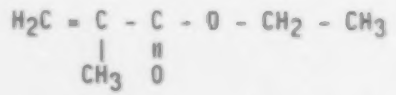
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
BOD	Biological oxygen demand
BODT	Biochemical oxygen demand, theoretical
EKG	Electrocardiogram
i.p.	Intraperitoneal
K_{oc}	Soil sorption coefficient
K_{ow}	Octanol/water partition coefficient
LD_{50}	Dose lethal to 50% of recipients
MED	Minimum effective dose
P	Partition coefficient
ppm	Parts per million
RQ	Reportable quantity
UV	Ultraviolet
WS	Water solubility

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Ethyl methacrylate is the common chemical name, but this compound is also known by the following synonyms: 2-propenoic, 2-methyl-, ethyl ester (current CAS usage); ethyl 2-methylate; ethyl 2-methyl-2-propenoate; methacrylic acid, ethyl ester; ethyl methyl acrylate. The structure, molecular weight, empirical formula and CAS Registry number for ethyl methacrylate are as follows:



Molecular weight: 114.14

Empirical formula: $C_6H_{10}O_2$

CAS Registry number: 97-63-2

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Ethyl methacrylate is a colorless liquid with an acrid acrylate odor (U.S. EPA, 1985a). It is soluble in alcohol and ether (Weast, 1985). Selected physical properties are listed below:

Melting point:	<-50°C	U.S. EPA, 1985a
Boiling point:	117°C	U.S. EPA, 1985a
Specific gravity (0°C):	0.9135 (20/4°C)	Weast, 1985
Refractive index:	1.4147 (20°C)	Weast, 1985
Water solubility: at 20°C	5600 ppm	U.S. EPA, 1985a
Vapor pressure mm Hg: at 20°C	14 mm Hg	U.S. EPA, 1985a
Log K_{ow}:	1.94	Hansch and Leo, 1985
Vapor density:	3.94 (16°C)	U.S. EPA, 1985a
Flash point (open cup):	21.1°C	Hawley, 1981

Ethyl methacrylate is readily polymerized (Hawley, 1981); in the absence of sufficient inhibitor or when subjected to elevated temperatures, a violent polymerization reaction may occur (U.S. EPA, 1985a). Commercial grades of ethyl methacrylate typically contain 22-28 ppm of hydroquinone or 15-20 ppm of the methyl ether of hydroquinone as an inhibitor (U.S. EPA, 1985a; Nemec and Kirch, 1981).

The methacrylic acid esters are chemically reactive compounds and can undergo reactions at the terminal vinylic carbon, at the double bond, at the allylic methyl group, at the ester moiety and at the functional groups in the nonmethacrylate moiety (Nemec and Kirch, 1981).

1.3. PRODUCTION DATA

U.S. EPA (1977) cites Rohm and Haas Co. (Philadelphia) as an importer of 1-10 million pounds of ethyl methacrylate and DuPont (Belle, WV) as a manufacturer whose production reports are confidential. SRI (1985) lists DuPont (Belle, WV) as the only U.S. manufacturer of ethyl methacrylate. Total consumption from all sources is probably in multimillions of pounds annually, based on the use of ethyl methacrylate.

Ethyl methacrylate is produced by the reaction of methacrylic acid or methyl methacrylate with ethyl alcohol (Hawley, 1981). Current commercial production of methyl methacrylate-methacrylic acid involves converting acetone cyanohydrin to methacrylamide sulfate, which is hydrolyzed and esterified to the methacrylate ester (Nemec and Kirch, 1981).

1.4. USE DATA

Ethyl methacrylate is primarily used in the production of acrylic polymers, but is also used as a chemical intermediate (U.S. EPA, 1985a). The acrylic polymers are used in paints and coatings and in components for the automotive, aerospace and furniture industries (U.S. EPA, 1985a). Ethyl

methacrylate polymers are used in dentistry for dentures, plates, and cements (Freeman, 1965; U.S. EPA, 1985a) and in the production of contact lenses (Refojo, 1979) and artificial fingernails (Lee and Orlowski, 1976).

1.5. SUMMARY

Ethyl methacrylate is a colorless liquid with an acrid acrylate odor (U.S. EPA, 1985a). It is chemically reactive and can be readily polymerized (Nemec and Kirch, 1981; Hawley, 1981). The primary use of ethyl methacrylate is for the production of acrylic polymers for use in building automotive, aerospace and furniture components and in the manufacture of paint and coatings (U.S. EPA, 1985a). Ethyl methacrylate polymers are also used for making dentures (Freeman, 1965), contact lenses (Refojo, 1979) and artificial fingernails (Lee and Orlowski, 1976). Domestic production figures are not available for ethyl methacrylate; however, its uses suggest that multimillion pound quantities are produced annually. DuPont (Belle, WV) is the sole U.S. manufacturer (SRI, 1985). In 1977, Rohm and Haas Co. imported between 1 and 10 million pounds of ethyl methacrylate (U.S. EPA, 1977).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

Pertinent data regarding the environmental fate processes for ethyl methacrylate could not be located in the available literature as cited in the Appendix. However, the environmental fate of ethyl methacrylate has been estimated from its physical properties and chemical structure and by analogy to the environmental fate processes and experimental data of similar compounds such as ethyl acrylate and various methacrylic acid esters.

2.1. WATER

2.1.1. Hydrolysis. Aliphatic acid esters are susceptible to hydrolysis (Mabey and Mill, 1978), suggesting that ethyl methacrylate may hydrolyze in water; however, the rate of hydrolysis can only be determined by experimental data. At pH 7 and 25°C, ethyl acrylate has a hydrolytic half-life of 3.5 years (Mabey and Mill, 1978); therefore, by analogy to ethyl acrylate, ethyl methacrylate would not be expected to hydrolyze significantly in water.

2.1.2. Oxidation. Pertinent data regarding the oxidation of ethyl methacrylate could not be located in the available literature as cited in the Appendix.

2.1.3. Photolysis. In cyclohexane and methanol solvents, the ethyl and methyl esters of acrylic and methacrylic acids do not absorb light strongly in the sunlight region of the UV spectrum (Brunn et al., 1976). Assuming similar absorption, ethyl methacrylate is not expected to be significantly degraded by direct photolysis in water.

2.1.4. Microbial Degradation. Methyl methacrylate, methyl acrylate, ethyl acrylate and butyl acrylate have all been confirmed to be "well-biodegradable" by the Japanese MITI test, indicating that oxygen consumption exceeded 30% of theoretical after 2 weeks of incubation using an activated sludge seed (Sasaki, 1978).

Price et al. (1974) examined the biodegradability of ethyl acrylate in BOD tests. Using an unacclimated seed, 5- and 20-day BODTs of 28 and 35% were determined, respectively; however, using an acclimated seed, 5- and 20-day BODTs of 66 and 79% were found, respectively. Ekhina and Ampleeva (1977) noted that methyl methacrylate, butyl methacrylate, methyl acrylate and ethyl acrylate all created a considerable oxygen demand during 5-, 15- and 20-day BOD tests.

Ethyl acrylate has also been found to be metabolized by methane-enriched cultures and amenable to anaerobic biotechnology (Chou et al., 1979; Speece, 1983).

Analogy of the above data to ethyl methacrylate suggests that ethyl methacrylate is biodegradable; however, the rate or significance of ethyl methacrylate biodegradation in natural waters cannot be predicted without additional data.

2.1.5. Volatilization. Estimation of Henry's Law Constant at 20°C from the ratio of vapor pressure (14 mm Hg) to WS (5600 ppm) yields a value of 3.8×10^{-4} atm·m³/mol. Estimation of Henry's Law Constant from the chemical structure (both bond and group contribution) method of Hine and Mookerjee (1975) yields a value of ~10^{-4} atm·m³/mol. These values of Henry's Law Constant indicate that volatilization is significant from all environmental waters (Lyman et al., 1982). The volatilization half-life of ethyl methacrylate from a body of water 1 m deep, with a water current speed of 1 m/sec and wind velocity of 3 m/sec, can be calculated to be 5.6 hours from the Henry's Law Constant of 3.8×10^{-4} atm·m³/mol using the method of Lyman et al. (1982).

2.1.6. Adsorption. As noted in Section 2.3.2., ethyl methacrylate is not expected to be tightly adsorbed to soil. Therefore, adsorption to particulate organic matter and sediment in water is probably not important.

2.1.7. Bioaccumulation. The following two equations are recommended for estimating the BCF of an organic compound (Lyman et al., 1982):

$$\log BCF = 0.76 \log K_{ow} - 0.23 \quad (2\text{-}1)$$

$$\log BCF = 2.791 - 0.564 \log WS \text{ (in ppm)} \quad (2\text{-}2)$$

The BCF values calculated from Equations (2-1) and (2-2) for ethyl methacrylate (log K_{ow} of 1.94; WS of 5600 ppm) are 17.6 and 4.8, respectively. These values suggest that bioaccumulation of ethyl methacrylate in aquatic organisms is not very significant.

2.2. AIR

The rate constants for the atmospheric reaction of ethyl methacrylate with hydroxyl radicals and with ozone have been theoretically calculated to be 5×10^{-11} cm³/molecule-sec (HO•) and 1.3×10^{-17} cm³/molecule-sec (O_3) (U.S. EPA, 1985b). Assuming average atmospheric concentrations of 8×10^5 HO• molecules/cm³ and 6×10^{11} O_3 molecules/cm³, the half-life of ethyl methacrylate (with respect to the combined effect of HO• and O_3) has been calculated to be 3.99 hours (U.S. EPA, 1985b). The half-lives with respect to only HO• or O_3 can be calculated to be 4.8 and 24.7 hours, respectively.

Direct photolysis is not expected to be significant, since ethyl methacrylate does not absorb light strongly in the sunlight region of the UV spectrum (Brunn et al., 1976).

2.3. SOIL

2.3.1. Microbial Degradation. Pertinent data regarding the microbial degradation of ethyl methacrylate in soil could not be located in the available literature as cited in the Appendix.

2.3.2. Adsorption. K_{oc} values can be estimated using the following two regression equations (Lyman et al., 1982):

$$\log K_{oc} = 3.64 - 0.55 \log WS \text{ (in ppm)} \quad (2\text{-}3)$$

$$\log K_{oc} = 0.544 \log K_{ow} + 1.377 \quad (2\text{-}4)$$

The K_{oc} values calculated from Equations (2-3) and (2-4) for ethyl methacrylate (log K_{ow} of 1.94; WS of 5600 ppm) are 38 and 270, respectively. Estimation of K_{oc} by the molecular topology and quantitative structure-activity relationship analysis method of Sabljic (1984) yields a K_{oc} value of 298. The estimated K_{oc} values of 270 and 298 predict medium soil mobility, while the K_{oc} value of 38 predicts very high soil mobility (Swann et al., 1983). In either case, ethyl methacrylate appears susceptible to significant leaching in soil.

2.3.3. Volatilization. The vapor pressure of ethyl methacrylate (14 mm Hg at 20°C) indicates that it will volatilize from dry surfaces quite rapidly. In moist soils, volatilization may be significant since ethyl methacrylate is expected to significantly volatilize from water (see Section 2.1.5.).

2.4. SUMMARY

Experimental data pertaining to the environmental fate processes for ethyl methacrylate were not found; therefore, its environmental fate has been estimated from its physical properties, chemical structure and by analogy to the fate of similarly-structured compounds. In water, hydrolysis, direct photolysis, adsorption and bioaccumulation are not expected to be important. Volatilization from water is expected to be significant, based on the calculated Henry's Law Constant. Ethyl, methyl and butyl acrylate, as well as methyl methacrylate, have been shown to be significantly biodegradable (Sasaki, 1978; Price et al., 1974; Ekhina and Ampleeva, 1977). By analogy, ethyl methacrylate is expected to be biodegradable.

Once released to the atmosphere, ethyl methacrylate is expected to react rapidly with hydroxyl radicals and ozone, with a calculated half-life of ~4 hours (U.S. EPA, 1985b). Ethyl methacrylate is estimated to be moderately to highly mobile in soil, and therefore susceptible to significant leaching. It may also volatilize significantly from soil surfaces.

3. EXPOSURE

3.1. WATER

No aquatic monitoring data were found for ethyl methacrylate in the available literature as cited in the Appendix. The U.S. EPA STORET Data Base contained no postings for the compound.

Ethyl methacrylate may be discharged with waste- water generated by manufacturing and use operations or through the leaching of unreacted ethyl methacrylate monomer from polymer products that have been landfilled.

3.2. FOOD

Pertinent monitoring data regarding ethyl methacrylate in food could not be located in the available literature as cited in the Appendix. It was not determined if ethyl methacrylate polymer products are used in food storage containers or in food wrapping; however, if they are used, any unreacted monomer may leach into foods contacting the polymer.

3.3. INHALATION

Pertinent monitoring data for ethyl methacrylate in ambient air could not be located in the available literature as cited in the Appendix; however, atmospheric emissions may result from its manufacture and use.

According to the preliminary results (as of September 20, 1985) of the National Occupational Exposure Survey, the number of American employees potentially exposed to ethyl methacrylate is 2520 (NIOSH, 1985).

The high temperature degradation of polydimethacrylates can result in the formation of ethyl methacrylate (Lomakin et al., 1984), suggesting that incineration of methacrylate polymers may produce ethyl methacrylate as a combustion product, which may in turn be released to the atmosphere through stack emissions.

3.4. DERMAL

Pertinent data regarding dermal exposure could not be located in the available literature as cited in the Appendix. Exposure potential may be greatest in occupational settings.

3.5. SUMMARY

Monitoring data regarding exposure to ethyl methacrylate through food, dermal exposure, ambient water or ambient air could not be located in the available literature. Ethyl methacrylate may be emitted to the atmosphere or to water from discharges generated during its industrial manufacture or use. Leaching of the unreacted monomer from polymers made from ethyl methacrylate may be a potential source of exposure to either groundwaters beneath landfills that contain the polymers, or to foods packaged in the polymer products. Incineration of polydimethacrylates may release ethyl methacrylate into the atmosphere.

4. PHARMACOKINETICS

Pertinent data regarding the absorption, distribution, metabolism or excretion of ethyl methacrylate could not be located in the available literature as cited in the Appendix.

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of ethyl methacrylate could not be located in the available literature as cited in the Appendix.

5.2. MUTAGENICITY

Ethyl methacrylate was not mutagenic in the plate incorporation assay using Salmonella typhimurium strains TA1535, TA1537, TA1538, TA98 or TA100, with or without metabolic activation. The concentration of ethyl methacrylate was 40-2500 µg/plate (Waegemaekers and Bensink, 1984).

5.3. TERATOGENICITY

Groups of five Sprague-Dawley rats were injected i.p. on days 5, 10 and 15 of gestation with 0.1223, 0.2446 or 0.4076 mL/kg of ethyl methacrylate, or 111.7, 223.4 or 372.3 mg/kg/day, respectively (assuming the density of ethyl methacrylate is 0.9135 g/mL) (Singh et al., 1972). Control groups of five pregnant Sprague-Dawley rats followed the same protocol, but with 0.82 mL/kg of distilled water, normal saline or cottonseed oil. A fourth control group was composed of five pregnant Sprague-Dawley rats that received no treatment. All rats were killed on day 20 of gestation. As summarized in Table 5-1, the number of fetuses or the incidence of skeletal abnormalities was not adversely affected. There was a significant decrease in the mean body weight of the fetuses in the ethyl methacrylate-treated group when compared with the untreated control group, but not when compared with the distilled water, normal saline or cottonseed oil control groups. There was an increased number of resorptions in all the ethyl methacrylate-treated rats when compared with the pooled control group in which the resorption rate was between 1.3 and 7.5% (95% confidence interval). There

TABLE 5-1

Teratogenicity of Ethyl Methacrylate Given Intraperitoneally to Pregnant Rats on Days 5, 10 and 15 of Gestation*

Groups	Number of Fetuses	Mean Fetal Weight	Resorptions	Gross Abnormalities	Skeletal Abnormalities
Untreated control	59	4.83±0.01	0	0	0
Distilled water	36	3.82±0.12	3 (7.7%)	0	1
Normal saline	50	4.15±0.11	4 (7.4%)	1	2
Cottonseed oil	50	3.85±0.08	0	1	4
Ethyl methacrylate (0.1223 mt/kg)	53	4.26±0.36	5 (9.4%)	3	1
Ethyl methacrylate (0.2446 mt/kg)	53	3.30±0.37	6 (12.5%)	5	2
Ethyl methacrylate (0.4076 mt/kg)	58	3.80±0.08	7 (12.1%)	8	3

*Source: Singh et al., 1972

was a statistically significant increase in the incidence of gross abnormalities in the fetuses from treated rats. In the high-dose groups, there were eight fetuses with gross abnormalities, including five with hemangiomas (one on a front leg, one on a hind leg and three on the shoulder), one with twisted hind legs, one without a tail and one smaller than the others with a compacted head and neck. In the middle-dose group, five fetuses had hemangiomas in the neck region. In the low-dose group, two fetuses had hemangiomas in the shoulders and one had twisted hind legs.

5.4. REPRODUCTIVE EFFECTS

Pertinent data regarding the reproductive effects of ethyl methacrylate could not be located in the available literature as cited in the Appendix.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Pertinent data regarding the chronic or subchronic toxicity of ethyl methacrylate could not be located in the available literature as cited in the Appendix.

5.6. OTHER RELEVANT INFORMATION

According to Deichman (1941), oral administration of toxic doses of methyl, ethyl and N-butyl methacrylate to rats and rabbits resulted in an initially increased breathing rate (within 2-5 minutes), followed by decreased breathing rate (within 15-40 minutes), motor weakness and gastric epithelial corrosion. In the rats, lacrimation accompanied the increased breathing rate and hematuria occurred. Death appeared to result from respiratory failure accompanied by dyspnea, loss of reflex activity, increased urination and defecation and coma. As indicated in Table 5-2, the LD_{50} in rats for ethyl methacrylate is almost twice the LD_{50} for methyl methacrylate.

TABLE 5-2

Acute Toxicity (LD_{50}) of Ethyl Methacrylate, Methyl Methacrylate and Ethyl Acrylate

Chemical	Molecular Weight[a]	Density[a] (mg/mℓ)	Log p[b]	Route	Species	LD_{50} (mg/kg)	Reference
Ethyl methacrylate	114.15	913.5	NR	oral	rat	13,519.8	Deichman, 1941
Ethyl methacrylate	114.15	913.5	1.94	oral	mouse	7,835.3	Tanii and Hashimoto, 1982
Ethyl methacrylate	114.15	913.5	NR	i.p.	rat	1,117.0	Singh et al., 1972
Ethyl methacrylate	114.15	913.5	NR	i.p.	mouse	1,250.58	Autian, 1975
Methyl methacrylate	100.13	944.0	NR	oral	rat	7,939.0	Deichman, 1941
Methyl methacrylate	100.13	944.0	1.38	oral	mouse	5,203.76	Tanii and Hashimoto, 1982
Methyl methacrylate	100.13	944.0	NR	i.p.	rat	1,253.6	Singh et al., 1972
Methyl methacrylate	100.13	944.0	NR	i.p.	mouse	1,130.9	Autian, 1975
Ethyl acrylate	100.13	923.4	NR	oral	rat	760	U.S. EPA, 1981
Ethyl acrylate	100.13	923.4	1.33	oral	mouse	1,799.3	Tanii and Hashimoto, 1982
Ethyl acrylate	100.13	923.4	NR	i.p.	mouse	598.4	Autian, 1975

[a]Source: Weast, 1985

[b]p = partition coefficient

NR = Not reported

Groups of 25 male and 25 female Wistar rats were exposed 50 0, 6-7, 60-70 or 2000 ppm of methyl methacrylate in their drinking water for 2 years (Borzelleca et al., 1964). The dose levels of methyl methacrylate, based on experimental data for body weight and fluid consumption, were 0, 0.55, 5.39 or 133 mg/kg/day for males and 0, 0.753, 7.53 or 179 mg/kg/day for females, respectively. The endpoints evaluated included body and organ weights, mortality, hematology, urinalysis and histological evaluation. The only significant effect reported was an increased relative kidney weight in the high-dose female rats. Motoc et al. (1971) reported unspecified kidney lesions, dystrophic and necrotic liver lesions and gastric ulcers in rats treated by gavage twice a week for 3, 5 or 8 months with ~115 mg/kg/day methyl methacrylate.

Groups of 30 pregnant rats were exposed to 0, 100 or 1000 ppm (0, 409, 4094 mg/m^3) of methyl methacrylate for 5 hours/day on days 6 through 15 of gestation (Hodge and Palmer, 1977). An increased incidence of resorptions was reported at the 1000 ppm exposure level, but no adverse effects were reported at $\leq$100 ppm. Groups of five pregnant Sprague-Dawley rats were injected on days 5, 10 and 15 of gestation with 0, 0.1328, 0.2656 or 0.4427 mℓ/kg of methyl methacrylate (Singh et al., 1972). At dose levels $\geq$0.1328 mℓ/kg (125.36 mg/kg) teratogenic and fetotoxic effects were reported.

Inhalation of both methyl methacrylate and ethyl methacrylate by mice increased the length of sodium phenobarbital-induced sleeping time, indicating an effect on the hepatic enzyme system (Lawrence and Autian, 1972). Despite the differences in the exposure level and time, methyl methacrylate appeared to have a more potent effect than ethyl methacrylate.

As reviewed by Autian (1975), methyl methacrylate and ethyl methacrylate (along with other methacrylate monomers) irreversibly reduced the contraction rate of the isolated, perfused rabbit heart and increased the coronary flow (Mir et al., 1973a); inhibited the spontaneous contractions of guinea pig ileum *in vitro* (Mir et al., 1973b); and affected the blood pressure, heart rate, EKG and respiration of anesthetized dogs treated intravenously (Mir et al., 1974). In dogs, both methyl and ethyl methacrylate initially caused decreased blood pressure followed by a secondary rise, decreased the heart and respiration rate and similarly altered the EKG, which changed with increasing dose levels (Mir et al., 1974).

Autian (1975) suggested that the low toxicity of the acrylic and methacrylic monomers may be due to their rapid metabolism and elimination, but did not provide specific substantiating data. Application of mathematical models (Free-Wilson and Hansch) indicated that the acrylic acid monomers might undergo hydrolysis, producing the more toxic acrylic acid moiety, but that the toxicity of the methacrylic acid monomers might be more directly related to the intact molecule (Autian, 1975). A positive relationship was reported between the partition coefficients of the various acrylic and methacrylic monomers and their acute toxicities, as indicated by the LD_{50} values (Autian, 1975). Tanii and Hashimoto (1982) reported that the acute toxicity (as indicated by LD_{50} values) of the acrylic and methacrylic monomers correlated best with the lipophilicity of the acrylic and methacrylic monomers as indicated by the partition coefficient. As shown in Table 5-2, ethyl acrylate had the lowest LD_{50} and the lowest log P, ethyl methacrylate had the highest LD_{50} and the highest log P, and methyl methacrylate was in the middle. More recently, Dillingham et al. (1983) reported that lipophilicity, *in vitro* hemolytic activity and long-term *in vivo* toxicity may be closely related in the acrylate and methacrylate esters.

5.7. SUMMARY

Pertinent data regarding the carcinogenicity, reproductive effects, subchronic and chronic effects of ethyl methacrylate could not be located in the available literature as cited in the Appendix. Ethyl methacrylate was not mutagenic in Salmonella typhimurium with or without metabolic activation (Waegemaekers and Bensink, 1984). Ethyl methacrylate caused a significantly increased incidence in gross abnormalities after i.p. administration in rats at all dose levels tested (Singh et al., 1972). Acute oral exposure to ethyl methacrylate resulted in an increased and then decreased breathing rate, dyspnea, coma and death due to respiratory failure (Deichman, 1941).

6. AQUATIC TOXICITY

6.1. ACUTE

Little information concerning the effects of ethyl methacrylate on aquatic organisms could be located in the available literature. Bringmann et al. (1980) reported that 178 mg/ℓ caused a 5% decrease in cell count of a Chilomonas paramecium culture.

6.2. OTHER RELEVANT INFORMATION

Pertinent data regarding chronic toxicity. effects on aquatic plants, and residues of ethyl methacrylate in aquatic organisms could not be located in the available literature as cited in the Appendix.

6.3. SUMMARY

The only information about aquatic toxicity of ethyl methacrylate was provided by Bringmann et al. (1980), who reported a toxicity threshold of 178 mg/ℓ for Chilomonas paramecium.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Pertinent guidelines and standards, including EPA ambient water and air quality criteria, drinking water standards, FAO/WHO/ADIs, EPA or FDA tolerances for raw agricultural commodities or foods, and ACGIH, NIOSH or OSHA occupational exposure limits could not be located in the available literature as cited in the Appendix.

7.2. AQUATIC

Pertinent data regarding guidelines and standards for the protection of aquatic biota from the effects of ethyl methacrylate could not be located in the available literature as cited in the Appendix.

8. RISK ASSESSMENT

No information on the carcinogenicity, reproductive effects, subchronic or chronic toxicity of ethyl methacrylate is available. Ethyl methacrylate is not mutagenic to S. typhimurium with or without metabolic activation (Waegemaekers and Bensink, 1984). The only animal study of ethyl methacrylate is the teratogenicity study by Singh et al. (1972), in which rats were treated i.p. with 111.7, 223.4 or 372.3 mg/kg of ethyl methacrylate on days 5, 10 and 15 of pregnancy. A significant increase in the incidence of resorptions and in the incidence of gross abnormalities (primarily hemangiomas, but including twisted hind legs, tail agenesis and microcephaly) occurred in all groups treated with ethyl methacrylate. Ethyl methacrylate is toxic following oral as well as i.p. administration; however, 6-12 times the intraperitoneal dose is necessary to produce the same effects from oral administration. Therefore, an ADI based on an i.p. administered dose would be too low. On the other hand, the rats were treated on only 3/21 days of gestation, and continuous oral exposure during pregnancy may provide a similar total exposure dose as that provided during the Singh et al. (1972) study. In addition to the dose level, the timing of the exposure is important in teratogenicity studies. The predominant anomaly observed in the rats was hemangiomas primarily in the neck region. Although a hemangioma is an overgrowth of vascular tissue, it is often considered a developmental anomaly rather than a true neoplastic disease, especially when the hemangioma is present at birth. In humans, some kinds of hemangiomas may be associated with mental retardation, seizures, hemiplegia and radio opacities in the skull (Robbins et al., 1984). Some hemangiomas may be associated with brain stem or eye field anomalies (Robbins et al., 1984), as in the case of one rat fetus that had been exposed to methyl methacrylate.

Although there are insufficient data on ethyl methacrylate with which to derive an ADI, there are several studies which suggest that an ADI for ethyl methacrylate could be derived by analogy to methyl methacrylate (Deichman, 1941; Autian, 1975; Mir et al., 1973a, 1974; Tanii and Hashimoto, 1982). In addition to chemical similarities, the acute toxic effects of ethyl methacrylate and methyl methacrylate are similar. However, the oral LD_{50} in rats for ethyl methacrylate is almost twice the oral LD_{50} in rats for methyl methacrylate suggesting that ethyl methacrylate is less toxic by the oral route. Both ethyl methacrylate and methyl methacrylate produce similar teratogenic effects in rats following i.p. injections of 111.7 and 125 mg/kg, respectively (Singh et al., 1972). Methyl methacrylate also caused teratogenic effects after inhalation exposure to 4094 mg/m³, but not 409 mg/m³ for 5 hours/day on days 6 through 15 of gestation (Hodge and Palmer, 1977). By expanding these exposures over 24 hours, assuming a 50% absorption rate, multiplying by the rat inhalation rate of 0.223 m³/day and dividing by the reference weight of a rat (0.35 kg), an estimated oral NOAEL of 27.2 mg/kg/day, and a LOAEL of 272 mg/kg/day can be derived for the teratogenic effects of methyl methacrylate exposure on rats.

An ADI of 0.075 mg/kg/day for methyl methacrylate was derived by U.S. EPA (1985c) from the animal NOEL of 7.5 mg/kg/day in the rat drinking water study of Borzelleca et al. (1964), which is also protective for the adverse developmental effects of methyl methacrylate. Since methyl methacrylate and ethyl methacrylate produce adverse developmental effects at comparable dose levels following i.p. administration and also that ethyl methacrylate appears to be less toxic than methyl methacrylate following oral administration, an ADI for ethyl methacrylate may be derived by analogy to methyl methacrylate. Therefore, an ADI of 0.086 mg/kg/day for ethyl methacrylate

was calculated by multiplying the ADI for methyl methacrylate (0.075 mg/kg/day) by the molecular weight ratio of ethyl methacrylate to methyl methacrylate (114.5/100.13). The ADI of 0.086 mg/kg/day or 6.0 mg/day for a 70 kg human should be considered provisional until subchronic or chronic oral toxicity and oral teratogenicity studies are performed with ethyl methacrylate.

9. REPORTABLE QUANTITIES

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY

As indicated in Table 9-1, lack of oral or inhalation studies on the teratogenicity, reproductive or systemic effects of ethyl methacrylate after either subchronic or chronic exposure precludes derivation of an RQ.

9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY

Lack of carcinogenicity data precludes derivation of a potency factor, and classifies ethyl methacrylate as an IARC Group 3 and CAG (U.S. EPA, 1984) Group D compound.

TABLE 9-1

Ethyl Methacrylate

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:

Dose:

Effect:

Reference:

RV_d:

RV_e:

Composite Score:

RQ: Data are not sufficient for deriving an RQ

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APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

CASR online (U.S. EPA Chemical Activities Status Report)
CAS online STN International
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in October, 1986. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. Documentation of the Threshold Limit Values, 4th ed. (Includes Supplemental Documentation, 1981, 1982, 1983). Cincinnati, OH. 486 p.

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In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

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